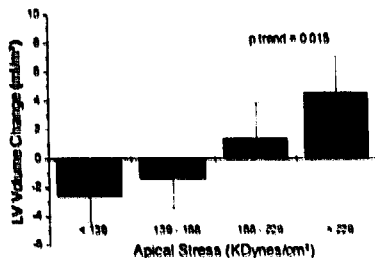


**Methods:** Utilizing finite element methods, we tested whether increased wall stress post MI predicts LV remodeling. 64 patients with acute anteroapical MI and high quality echo images were selected from the Healing and Early Afterload Reducing Therapy Trial, a trial of ACE inhibition following anterior MI. 31 patients received low dose (0.625 mg) ramipril and 33 received full dose (10 mg) ramipril. End-systolic LV models were constructed from orthogonal apical views obtained by echo two weeks post MI. LV wall stress, assessed by finite element methods, was compared with subsequent change in LV volume (14–80 days).

**Results:** LV volume change increased across quartiles of apical wall stress ( $p$ , trend = 0.015) in all patients, and in the low dose group ( $p$ , trend = 0.004), but was attenuated in the full dose group ( $p$ , trend = 0.72). Apical wall stress remained highly predictive of volume change after adjusting for infarct size and heart size in all patients ( $r = 0.58$ ;  $p = 0.03$ ) and in the low dose group ( $r = 0.71$ ;  $p = 0.004$ ).



**Conclusions:** Apical LV wall stress 2 weeks post MI predicts subsequent LV remodeling, and this relationship is attenuated by full dose ACE-inhibition, suggesting that ACE inhibition may modify the relationship between regional LV wall stress and remodeling.

2:15

#### 894-2 Sympathetic Denervation in Patients With Chronic Coronary Artery Disease: Relationship to Myocardial Perfusion and Metabolism

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Cardiac sympathetic denervation has been previously described in pts with coronary artery disease (CAD) following acute myocardial infarction, but there are little data correlating sympathetic innervation with perfusion and metabolism in patients with chronic coronary artery disease and ventricular dysfunction. PET imaging of myocardial perfusion (with nitrogen-13 ammonia, NH<sub>3</sub>), metabolism (with fluorine-18 fluorodeoxyglucose, FDG) and sympathetic innervation (with carbon-11 hydroxyephedrine, HED) was performed in 8 nondiabetic men, 54 ± 15 yrs, with known CAD referred for evaluation of myocardial ischemia and viability. A semi-automated program was used to determine NH<sub>3</sub> and FDG uptake and HED retention in 177 regions of interest encompassing the left ventricular (LV) myocardium. Perfusion, metabolism and innervation defects were defined as the percentage of LV with tracer uptake or retention > 2 SD below values obtained from a normal database. The extent of defects with each tracer was as follows (mean ± SD, range): NH<sub>3</sub> 37.3 ± 13.2% (21.7–61.1), FDG 44.2 ± 19% (19.9–75.6), and HED 36.5 ± 11.4 (18.1–51.8),  $p = NS$  for tracer effect.

**Conclusions:** In those patients with chronic CAD and ventricular dysfunction, the extent of sympathetic denervation matches the extent of ischemic injury and reduced glucose metabolism. Since the extent of sympathetic denervation exceeds the extent of ischemic injury acutely after MI, these results could be explained by sympathetic reinnervation during the recovery period.

2:30

#### 894-3 Comparison of Dobutamine Stress Tagged MRI With F-18 FDG PET to Assess Myocardial Viability in Patients With Chronic Ischemic Heart Disease

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**Background:** Characterizing the jeopardized but salvageable myocardium represents a key step in patient management after myocardial infarction. We evaluated regional strains in patients with chronic ischemic heart disease and compared myocardial viability assessed by strain measurements to viability assessed using FDG PET.

**Materials and Methods:** 14 patients with a history of myocardial infarction were referred to assess myocardial viability. Tagged breath-hold MRI (6

seconds) was acquired at rest, and under low-dose dobutamine infusion (10 µg/kg/min). Strain maps at rest and under stress were computed. FDG-PET images were acquired in 3D mode with a CTI/HR+ camera. Based on coronary angiography, clinical history and PET imaging, regions were assigned to one of the following groups: infarcted and non-viable regions (glucose uptake <5%), infarcted but viable regions (glucose uptake ≥70%), non-infarcted but ischemic regions, and normal regions.

**Results:** End-systolic maximum shortening (MS) remained severely depressed under stress ( $-1 \pm 6\%$ ) in infarcted regions, whereas angular deviation ranged from  $-30^\circ$  to  $50^\circ$ . When circumferential extent of infarcted regions with a glucose uptake <50% was of 2 or more segments, we observed larger end-systolic MS associated with large angular deviation that was corresponding to radial stretching. Maximum shortening was significantly larger ( $p < 0.05$ ) in all viable regions but remained inferior to normal regions (MS:  $-17 \pm 3\%$ , angle:  $-13 \pm 6^\circ$ ).

**Conclusion:** Tagged MR imaging and strain maps can differentiate non-viable and viable regions in patients with chronic ischemic heart disease.

2:45

#### 894-4 Differential Effects on Left Atrial Function of Pacing-Induced Ischemia in Patients With Diseased Proximal Left Anterior Descending and Proximal Left Circumflex Coronary Artery

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In left ventricular (LV) ischemia, compensatory augmentation of left atrial (LA) contraction enhances LV filling and performance, whereas loss of this atrial transport function exacerbates hemodynamic compromise. We hypothesized that one mechanism for the loss of this important enhancer of LV performance may be ischemic LA dysfunction. LA function was therefore compared in 8 patients with single-vessel left anterior descending (LAD, group A) and in 8 patients with single-vessel proximal left circumflex (LCx, group B) coronary artery stenosis at rest and immediately after pacing. LV and LA pressure-area relations were derived from simultaneous double-tip micromanometer pressure recordings and automatic boundary detection echocardiograms. Immediately after pacing, LV end-diastolic pressure rose from 8.8 to 18.7 mmHg in group A and from 9.4 to 19.5 mmHg in group B ( $p < 0.001$  vs rest for both groups), time constant of relaxation increased from 33.9 to 41.2 msec in group A ( $p < 0.01$  vs at rest) and from 34.2 to 55.6 msec in group B ( $p < 0.001$  vs rest), LA mean pressure rose from 9.3 to 19.2 mmHg in group A and from 10.4 to 19.2 mmHg in group B ( $p < 0.001$  vs rest for both groups) and LV stiffness increased from 0.098 to 0.202 cm<sup>-2</sup> in group A and from 0.096 to 0.194 mmHg in group B ( $p < 0.001$  vs rest for both groups). All these changes were similar in both groups. However, LV peak systolic pressure fell from 127.7 to 111.9 mmHg in group B while it remained unchanged in group A, LA systolic emptying index decreased from 0.26 to 0.15 in group B while it increased from 0.28 to 0.32 in group A ( $p < 0.001$  vs rest for both groups), LV stroke work index decreased from 1053.4 to 749.7 mmHg.cm<sup>2</sup> in group B ( $p < 0.05$ ) without a significant change in group A, the area of the LA loop of the LA pressure-area relation decreased from 14.4 to 9.3 mmHg.cm<sup>2</sup> in group B ( $p < 0.05$  vs rest) while it increased from 12.4 to 54.1 mmHg.cm<sup>2</sup> in group A ( $p < 0.01$  vs rest) and LA stiffness increased from 0.200 to 0.287 in group B ( $p < 0.001$  vs rest) without a significant change in group A.

**Conclusions:** In patients with LAD coronary artery stenosis, LV ischemia is associated with enhanced LA function, manifest as augmented LA A loops. However, in patients with proximal LCx coronary artery stenosis, LA branches might have been affected, rendering the LA ischemic and unable to increase its booster pump function. Ischemic depression of LA function contributes to further decrease of LV performance.

3:00

#### 894-5 Anterior Myocardial Ischemia Causes Simultaneous Left and Right Ventricular Contractile Dysfunction

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The contribution of the interventricular septum to global RV contractile function remains unknown. In order to examine the effects of septal and LV free wall ischaemia on LV and RV contractile performance, 8 open-chest, open pericardium pigs underwent mid LAD occlusion for 50 mins followed by 60 mins of reperfusion. A 6F integrated conductance catheter and micromanometer was inserted into both the LV and RV to assess simultaneous pressure and volume changes at baseline, during LAD occlusion, and at 15 and 60 mins of reperfusion. Ventricular volumes were calibrated from a transit time flow probe placed around the pulmonary artery and contractile indices were obtained from pressure-volume relations (PVR) during IVC occlusion.

## Results:

	RV EDV	LV EDV	RV ESV	LV ESV	RV ESP	LV ESP
Base	99.9	88.9	57.9	52.6	29.3	89.8
LAD ocl	112.8	102.7	64.9	61.4	30.8	91.2
P	0.013	0.024	0.018	0.014	0.48	0.7

EDV = end-diastolic volume (ml), ESV = end-systolic vol (ml), ESP = end-systolic pressure (mmHg).

	RVPRSW mmHg/ml	LVESPVR mmHg/ml
Baseline	19.0	1.1
15 mins reperfusion	13.7	0.71
60 mins reperfusion	16.8	1.14
P <sup>1</sup>	0.06	0.014

P<sup>1</sup> = comparison between baseline and 15 mins reperfusion. PRSW = preload recruitable stroke work, ESPVR end-systolic pressure-volume relations.

**Conclusions:** The increase in volumes with no increase in pressure generation during LAD occlusion suggests a fall in systolic contractile function in both ventricles. This persists at 15 mins reperfusion as shown by the fall in PRSW and ESPVR but returns to baseline by 60 mins. These results suggest that, even in the absence of a pericardium, the interventricular septum contributes significantly to global RV performance.

## 895 Quantitative Doppler Echocardiography

Wednesday, April 1, 1998, 2:00 p.m.-3:30 p.m.  
Georgia World Congress Center, Room 360W

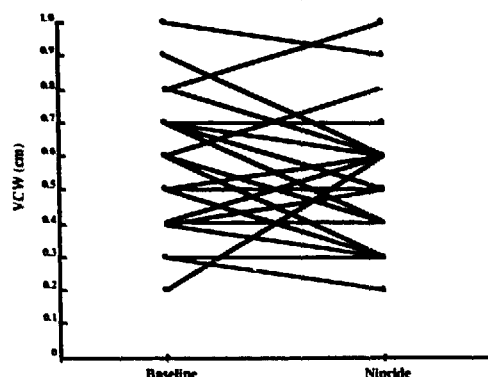
### 895-1 Effect of Afterload Reduction on Vena Contracta Width in Mitral Regurgitation

A.M. Kizilbash, D.L. Willett, M.E. Brickner, I. Afendi, S.K. Heinle, P.A. Grayburn. *University of Texas Southwestern and VA Medical Centers, Dallas, Texas*

**Background:** Vena contracta width (VCW) by Doppler color flow imaging has recently emerged as a simple marker of the severity of mitral regurgitation (MR). *In vitro* studies using a fixed orifice suggest that VCW is load independent. This study was done to assess the clinical effects of afterload reduction on VCW.

**Methods:** We studied 31 patients with chronic MR (21 men; 10 women, age 29 to 77 yrs) at baseline and during afterload reduction with sodium nitroprusside (nipride). Each patient had >15% reduction in systolic blood pressure during nipride (mean change  $148 \pm 27$  to  $115 \pm 25$  mmHg). VCW was measured at baseline and during nipride. All studies were read in random order by blinded observers.

**Results:** Mean values for VCW did not change from baseline to nipride ( $0.55 \pm 0.19$  vs  $0.50 \pm 0.19$ ,  $p = ns$ ). However, there was marked directional variability in individual pts such that VCW increased after nipride in 8 pts, decreased in 16 pts, and was unchanged in 7 pts (see figure).



**Conclusions:** 1) Vena contracta width is not load independent in individual patients with MR due to dynamic changes in the regurgitant orifice. 2) Acute afterload reduction worsens MR in some patients. 3) Nipride echocardiography may be helpful in patients with MR being considered for chronic vasodilator therapy to be sure that afterload reduction does not worsen the MR.

2:00

### 895-2 Hemisphere Versus Hemielipse: When is Each Most Accurate for Proximal Isovelocity Calculation of Regurgitant Flows

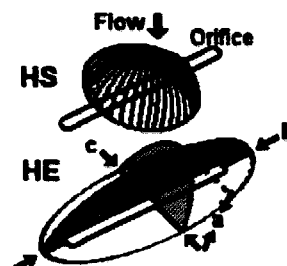
T. Buck, C.H.P. Jansen, A.P. Yoganathan, R.A. Levine, M.D. Handschumacher. *Massachusetts General Hospital, Boston, MA, USA*

Variable proximal isovelocity surface area (PISA) shape has been a major obstacle to clinical calculation of regurgitant flow rate (Q). Two proposed approaches to improve accuracy have not been compared: 1) Analysis of centerline velocities (v's) to find the optimal v where a hemispherical (HS) shape works best; 2) a flexible hemieliptical (HE) formula applied to 2 perpendicular apical views. These were compared *in vitro* using circular (CIRC) and slit orifices (3:1, 8:1) of 0.2-0.8 cm<sup>2</sup> areas at 7 flows (20-140 ml/s) and 8 Nyquists (504 stages).

**Results:** 1) For CIRC orifices, the HS formula was highly accurate at the optimal v; the HE agreed with actual Qa over a range of v's, with slight underestimation because of flow oblique to the Doppler beam. 2) With increasing slit eccentricity, the HS progressively underestimated Q compared to the HE. 3) HE values could be empirically corrected for Doppler underestimation based on PISA geometry (a x b/c).

% errors: (Bold: No signif. underestimation)

Shape	Size	HS	HE	HEcorr
CIRC	0.2	-2.4 ± 3.8	-6.1 ± 1.9	0.05 ± 1.1
CIRC	0.8	-1.3 ± 2.5	-6.5 ± 1.9	1.91 ± 0.4
SLIT	0.2	-21.1 ± 12.3	-13.5 ± 1.4	-1.3 ± 0.3
SLIT	0.8	-50.6 ± 6.7	-14.5 ± 1.3	-1.68 ± 0.8



**Conclusion:** For a CIRC orifice (symmetric PISA in apical views), the HS optimal v approach is highly accurate. However, the hemielipse is most accurate over a broad range of orifice shapes and sizes, flows and Nyquists, and can readily be corrected for the Doppler angle effect.

2:30

### 895-3 Quantifying Mitral Regurgitation Using Correlated Doppler Measurements

P.W. Wilkerson, A.P. Yoganathan, R.A. Levine<sup>1</sup>. *Georgia Institute of Technology, Atlanta, Georgia, USA; <sup>1</sup>Massachusetts General Hospital, Boston, Massachusetts, USA*

**Background:** The hemi-ellipsoidal PISA technique requires three measured dimensions. The use of 2D Doppler yields images of the Doppler velocity flow field instead of the actual velocity field and hence inaccurate dimensions of the hemi-ellipsoidal contours. A technique to easily obtain the necessary hemi-elliptic dimensions to calculate the MR accurately from the Doppler images is needed.

**Methods:** Computational simulations were conducted to obtain the true and Doppler velocity flow fields proximal to the mitral regurgitant orifice, using symmetric (4-9 mm diameter) and slit-like (2:1-18:1 aspect ratio) orifices in the presence of aortic outflow. The three necessary hemi-elliptic dimensions were measured from both the true and Doppler flow fields. Ratios of the Doppler and true dimensions were analyzed with the PISA calculated MR from both flow fields to develop a set of correlation factors to apply to the Doppler measurements. *In vitro* studies were conducted to validate the set of correlation factors.

**Results:** The correlation factors developed only utilize the isovelocity value and measured Doppler dimensions. The factors are applied to the Doppler dimensions through a computer program, resulting in a new set of correlated Doppler dimensions, used to calculate the MR. The set of correlation factors only uses the three Doppler measurements and velocity as input to calculate the MR. Using the correlation factors for the computational simulations, the MR were predicted within  $2.1 \pm 6.5\%$  of the actual MR. The *in vitro* experiments validated the correlation factors, yielding calculated MR within 10% of the actual MR.